FORM LG-4878

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ESTABLISHED 1802
E. I. DU PONT DE NEMOURS & COMPANY
INCORPORATED
WILMINGTON, DELAWARE 19898

LEGAL DEPARTMENT

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September 11, 1992

Document Processing Center (TS-790)
Office of Pollution Prevention and Toxics
Environmental Protection Agency
401 M Street., S.W.
Washington, D.C. 20460
Attn: Section 8(e) Coordinator (CAP Agreement)

88920010975

Dear Coordinator:

8ECAP-0025

On behalf of the Regulatee and pursuant to Unit II B.1.b. and Unit II C of the 6/28/91CAP Agreement, E.I. Du Pont de Nemours and Co. hereby submits (*in triplicate*) the attached studies. Submission of this information is voluntary and is occasioned by unilateral changes in EPA's standard as to what EPA now considers as reportable information. Regulatee's submission of information is made solely in response to the new EPA §8(e) reporting standards and is not an admission: (1) of TSCA violation or liability; (2) that Regulatee's activities with the study compounds reasonably support a conclusion of substantial health or environmental risk or (3) that the studies themselves reasonably support a conclusion of substantial health or environmental risk.

The "Reporting Guide" creates new TSCA 8(e) reporting criteria which were not previously announced by EPA in its 1978 Statement of Interpretation and Enforcement Policy, 43 Fed Reg 11110 (March 16, 1978). The "Reporting Guide states criteria which expands upon and conflicts with the 1978 Statement of Interpretation. Absent amendment of the Statement of Interpretation, the informal issuance of the "Reporting Guide" raises significant due processes issues and clouds the appropriate reporting standard by which regulated persons can assure TSCA Section 8(e) compliance.

For Regulatee,

ORIGINAL

Mark H. Christman
Counsel
Legal D-7158
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Wilmington, DE 19898
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8ECAP

RECEIVED 3/24/95

ATTACHMENT 1

Submission of information is made under the 6/28/91 CAP Agreement, Unit II. This submission is made voluntarily and is occasioned by recent changes in EPA's TSCA §8(e) reporting standard; such changes made, for the first time in 1991 and 1992 without prior notice and in violation of Regulatee's constitutional due process rights. Regulatee's submission of information under this changed standard is not a waiver of its due process rights; an admission of TSCA violation or liability, or an admission that Regulatee's activities with the study compounds reasonably support a conclusion of substantial risk to health or to the environment. Regulatee has historically relied in good faith upon the 1978 Statement of Interpretation and Enforcement Policy criteria for determining whether study information is reportable under TSCA §8(e), 43 Fed Reg 11110 (March 16, 1978). EPA has not, to date, amended this Statement of Interpretation.

After CAP registration, EPA provided the Regulatee the June 1, 1991 "TSCA Section 8(e) Reporting Guide". This "Guide" has been further amended by EPA, EPA letter, April 10, 1992. EPA has not indicated that the "Reporting Guide" or the April 1992 amendment supersedes the 1978 Statement of Interpretation. The "Reporting Guide" and April 1992 amendment substantively lowers the Statement of Interpretation 's TSCA §8(e) reporting standard[2]. This is particularly troublesome as the "Reporting Guide" states criteria, applied retroactively, which expands upon and conflicts with the Statement of Interpretation.[3] Absent amendment of the Statement of Interpretation, the informal issuance of the "Reporting Guide" and the April 1992 amendment clouds the appropriate standard by which regulated persons must assess information for purposes of TSCA §8(e).

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[2]In sharp contrast to the Agency's 1977 and 1978 actions to soliciting public comment on the proposed and final §8(e) Policy, EPA has unilaterally pronounced §8(e) substantive reporting criteria in the 1991 Section 8(e) Guide without public notice and comment, See 42 Fed Reg 45362 (9/9/77), "Notification of Substantial Risk under Section 8(e): Proposed Guidance".

[3]A comparison of the 1978 Statement of Interpretation and the 1992 "Reporting Guide" is a appended.

Throughout the CAP, EPA has mischaracterized the 1991 guidance as reflecting "longstanding" EPA policy concerning the standards by which toxicity information should be reviewed for purposes of §8(e) compliance. Regulatee recognizes that experience with the 1978 Statement of Interpretation may cause a review of its criteri. Regulatee supports and has no objection to the Agency's amending reporting criteria *provided that* such amendment is not applied to the regulated community in an unfair way. However, with the unilateral announcement of the CAP under the auspices of an OCM enforcement proceeding, EPA has wrought a terrific unfairness since much of the criteria EPA has espoused in the June 1991 Reporting Guide and in the Agency's April 2, 1992 amendment is new criteria which does not.exist in the 1978 Statement of Interpretation and Enforcement Policy.

The following examples of new criteria contained in the "Reporting Guide" that is not contained in the Statement of Interpretation follow:

o even though EPA expressly disclaims each "status report" as being preliminary evaluations that should not be regarded as final EPA policy or intent[4], the "Reporting Guide" gives the "status reports" great weight as "sound and adequate basis" from which to determine mandatory reporting obligations. ("Guide" at page 20).

o the "Reporting Guide" contains a matrix that establishes new numerical reporting "cutoff" concentrations for acute lethality information ("Guide" at p. 31). Neither this matrix nor the cutoff values therein are contained in the Statement of Interpretation. The regulated community was not made aware of these cutoff values prior to issuance of the "Reporting Guide" in June, 1991.

othe "Reporting Guide" states new specific definitional criteria with which the Agency, for the first time, defines as 'distinguishable neurotoxicological effects'; such criteria/guidance not expressed in the 1978 Statement of Interpretation.[5];

othe "Reporting Guide" provides new review/ reporting criteria for irritation and sensitization studies; such criteria not previously found in the 1978 Statement of Interpretation/Enforcement Policy.

othe "Reporting Guide" publicizes certain EPA Q/A criteria issued to the Monsanto Co. in 1989 which are not in the Statement of Interpretation; have never been published in the Federal Register or distributed by the EPA to the Regulatee. Such Q/A establishes new reporting criteria not previously found in the 1978 Statement of Interpretation/Enforcement Policy .

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[4]The 'status reports' address the significance, if any, of particular information reported to the Agency, rather than stating EPA's interpretation of §8(e) reporting criteria. In the infrequent instances in which the status reports contain discussion of reportability, the analysis is invariably quite limited, without substantial supporting scientific or legal rationale.

[5] *See, e.g,* 10/2/91 letter from Du Pont to EPA regarding the definition of 'serious and prolonged effects' as this term may relate to transient anesthetic effects observed at lethal levels; 10/1/91 letter from the American Petroleum Institute to EPA regarding clarification of the Reporting Guide criteria.

In discharging its responsibilities, an administrative agency must give the regulated community fair and adequate warning to as what constitutes noncompliance for which penalties may be assessed.

> Among the myriad applications of the due process clause is the fundamental principle that statutes and regulations which purport to govern conduct must give an adequate warning of what they command or forbid.... Even a regulation which governs purely economic or commercial activities, if its violation can engender penalties, must be so framed as to provide a constitutionally adequate warning to those whose activities are governed.

Diebold, Inc. v. Marshall, 585 F.2d 1327, 1335-36 (D.C. Cir. 1978). *See also*, Rollins Environemntal Services (NJ) Inc. v. U.S. Environmental Protection Agency, 937 F. 2d 649 (D.C. Cir. 1991).

While neither the are rules, This principle has been applied to hold that agency 'clarification', such as the Statement of Interpretation, the "Reporting Guide" nor the April 1992 amendments will not applied retroactively.

> ...a federal court will not retroactively apply an unforeseeable interpretation of an administrative regulation to the detriment of a regulated party on the theory that the post hoc interpretation asserted by the Agency is generally consistent with the policies underlying the Agency's regulatory program, when the semantic meaning of the regulations, as previously drafted and construed by the appropriate agency, does not support the interpretation which that agency urges upon the court.

Standard Oil Co. v. Federal Energy Administration, 453 F. Supp. 203, 240 (N.D. Ohio 1978), aff'd sub nom. Standard Oil Co. v. Department of Energy, 596 F.2d 1029 (Em. App. 1978):

The 1978 Statement of Interpretation does not provide adequate notice of, and indeed conflicts with, the Agency's current position at §8(e) requires reporting of all 'positive' toxicological findings without regard to an assessment of their relevance to human health. In accordance with the statute, EPA's 1978 Statement of Interpretation requires the regulated community to use scientific judgment to evaluate the significance of toxicological findings and to determining whether they reasonably support a conclusion of a substantial risk. Part V of the Statement of Interpretation urges persons to consider "the fact or probability" of an effect's occurrence. Similarly, the 1978 Statement of Interpretation stresses that an animal study is reportable only when "it contains reliable evidence ascribing the effect to the chemical." 43 Fed Reg. at 11112. Moreover, EPA's Statement of Interpretation defines the substantiality of risk as a function of both the seriousness of the effect and the probability of its occurrence. 43 Fed Reg 11110 (1978). Earlier Agency interpretation also emphasized the "substantial" nature of a §8(e) determination. See 42 Fed Reg 45362, 45363

(1977). [Section 8(e) findings require "extraordinary exposure to a chemical substance...which critically imperil human health or the environment"].

The recently issued "Reporting Guide" and April 1992 Amendment guidance requires reporting beyond and inconsistent with that required by the Statement of Interpretation. Given the statute and the Statement of Interpretation's explicit focus on substantial human or environmental risk, whether a substance poses a "substantial risk" of injury requires the application of scientific judgment to the available data on a case-by-case basis.

If an overall weight-of-evidence analysis indicates that this classification is unwarranted, reporting should be unnecessary under §8(e) because the available data will not "reasonably support the conclusion" that the chemical presents a substantial risk of serious adverse consequences to human health.

Neither the legislative history of §8(e) nor the plain meaning of the statute support EPA's recent lowering of the reporting threshold that TSCA §8(e) was intended to be a sweeping information gathering mechanism. In introducing the new version of the toxic substances legislation, Representative Eckhart included for the record discussion of the specific changes from the version of H. R. 10318 reported by the Consumer Protection and Finance Subcommittee in December 1975. One of these changes was to modify the standard for reporting under §8(e). The standard in the House version was changed from "causes or contributes to an unreasonable risk" to "causes or significantly contributes to a substantial risk". This particular change was one of several made in TSCA §8 to avoid placing an undue burden on the regulated community. The final changes to focus the scope of Section 8(e) were made in the version reported by the Conference Committee.

The word "substantial" means "considerable in importance, value, degree, amount or extent". Therefore, as generally understood, a "substantial risk" is one which will affect a considerable number of people or portion of the environment, will cause serious injury and is based on reasonably sound scientific analysis or data. Support for the interpretation can be found in a similar provision in the Consumer Product Safety Act. Section 15 of the CPSA defines a "substantial product hazard" to be:

> "a product defect which because of the pattern of defect, the number of defective products distributed in commerce, the severity of the risk, or otherwise, creates a substantial risk of injury to the public."

Similarly, EPA has interpreted the word 'substantial' as a quantitative measurement. Thus, a 'substantial risk' is a risk that can be quantified, *See,* 56 Fed Reg 32292, 32297 (7/15/91). Finally, since information pertinent to the exposure of humans or the environment to chemical substances or mixtures may be obtained by EPA through Sections 8(a) and 8(d) regardless of the degree of potential risk, §8(e) has specialized function. Consequently, information subject to §8(e) reporting should be of a type which would lead a reasonable man to conclude that some type action was required immediately to prevent injury to health or the environment.

Attachment

Comparison:

Reporting triggers found in the 1978 "Statement of Interpretation/ Enforcement Policy",43 Fed Reg 11110 (3/16/78) and the June 1991 *Section 8(e) Guide.*

TEST TYPE	1978 POLICY CRITERIA EXIST?	New 1991 GUIDE CRITERIA EXIST?
ACUTE LETHALITY		
Oral	N}	Y}
Dermal	N}	Y}
Inhalation (Vapors)	}[6]	}[7]
aerosol	N}	Y}
dusts/ particles	N}	Y}
SKIN IRRITATION	N	Y[8]
SKIN SENSITIZATION (ANIMALS)	N	Y[9]
EYE IRRITATION	N	Y[10]
SUBCHRONIC (ORAL/DERMAL/INHALATION)	N	Y[11]
REPRODUCTION STUDY	N	Y[12]
DEVELOPMENTAL TOX	Y[13]	Y[14]

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[6] 43 Fed Reg at 11114, comment 14:

"This policy statements directs the reporitng of specifiec effects when unknown to the Administrator. Many routine tests are based on a knowledge of toxicity associated with a chemicalL unknown effects occurring during such a range test may have to be reported if they are those of concern tot he Agency and if the information meets the criteria set forth in Parts V and VII."

[7] Guide at pp.22, 29-31.

[8] Guide at pp-34-36.

[9] Guide at pp-34-36.

[10] Guide at pp-34-36.

[11] Guide at pp-22; 36-37.

[12] Guide at pp-22

[13] 43 Fed Reg at 11112

"Birth Defects" listed.

[14] Guide at pp-22

NEUROTOXICITY	N	Y[15]
CARCINOGENICITY	Y[16]	Y[17]
MUTAGENICITY		
In Vitro	Y}[18]	Y} [19]
In Vivo	Y}	Y}
ENVIRONMENTAL		
Bioaccumulation	Y}	N
Bioconcentration	Y}[20]	N
Oct/water Part. Coeff.	Y}	N
Acute Fish	N	N
Acute Daphnia	N	N
Subchronic Fish	N	N
Subchronic Daphnia	N	N
Chronic Fish	N	N
AVIAN		
Acute	N	N
Reproductive	N	N
Reprodcutive	N	N

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[15] Guide at pp-23; 33-34.

[16] 43 Fed Reg at 11112
"Cancer" listed

[17] Guide at pp-21.

[18] 43 Fed Reg at 11112; 11115 at Comment 15
"Mutagenicity" listed/ *in vivo* vs *invitro* discussed; discussion of "Ames test".

[19] Guide at pp-23.

[20] 43 Fed Reg at 11112; 11115 at Comment 16.

CAS #115-10-6
Chem: Methane, oxybis
Title: Subacute Inhalation Study of Dimethyl Ester (DME) In Rats
Date: August 11, 1980
Summary of Effects: EC 50 for anesthtic effect = 5-8%v/v; uncoordinated.

FOR DU PONT USE ONLY

Copies to: S. N. Boyd, Jr. (2)
W. H. Darnell (1)
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J. L. Riggs (1)
R. Irelan (1)
Bruce Evans (1)

E. I. du Pont de Nemours and Company
Haskell Laboratory for Toxicology and Industrial Medicine
Elkton Road, Newark, Delaware 19711

HASKELL LABORATORY REPORT NO. 583-80 MR NO. 3536-001

Material Tested	Haskell No.	Other Codes/Synonyms
Methane, oxybis*	13,203-02 CAS Registry No. 115106	Product Code 3710 dimethyl ether (DME), wood ether

Study Initiated/Completed
10/15/79 - 11/19/79

Material Submitted by
Bruce Evans
CD&P Dept.
Chestnut Run

SUBACUTE INHALATION STUDY OF DIMETHYL ETHER (DME) IN RATS

Introduction: Previous work (HLR-847-80) has shown the 4 hr. LC50 of dimethyl ether (DME) in rats is 16.4% (164,000 ppm). Another study[1] has shown minor effects (e.g., reduction in liver weight) when rats were exposed to up to 2% DME for 30 weeks by inhalation. The present two-week study was undertaken at 2 concentrations, 1% and 5%. Five percent was chosen as the high dose in an attempt to demonstrate toxic effects.

Protocols and Procedures:

General Protocol: Three groups of 10 male albino ChR-CD® rats approximately 8-weeks old, weighing 235 to 293 g were exposed to atmospheres of DME in air. Rats were exposed in 20 l glass battery jars, 6 hrs/day, 5 days/week for 2 weeks. Prior to exposure all rats were held for a 10-day pretest period to insure suitability. Following the 10th exposure half of the rats were sacrificed for pathologic examination and half of the rats were held for a 2-week recovery period. Except during exposure, rats were housed in pairs in wire mesh stainless-steel cages with Purina® Rodent Chow #5002 and water available ad libitum. Rats were weighed and observed daily (except weekends) throughout the exposure and recovery periods. During the second exposure week the rats were weighed both pre- and post-exposure.

Safety Procedures: The explosive limits of DME in air are 3.4 to 27%. Because of the hazardous nature of conducting inhalation exposures at explosive concentrations, the following precautions were taken: Chamber volumes were kept as small as practical; handling of rats was minimized; all metal to metal contacts in the chamber set-up were eliminated; all work was done in a high velocity hood;

electrical equipment was kept to a minimum and placed outside of the hood; all equipment, including the chamber parts, was grounded to prevent buildup of static electricity; flash arrestors were used on the DME tank and between the chambers and the exhaust vacuum pump; the DME was diluted with air to below explosive concentrations, as soon as possible; and access to the area was restricted.

Generation Procedure: DME is a gas which was received as a liquid under pressure. Atmospheres were generated by metering DME gas directly from one cylinder through two flow meters into the top of the chambers, where the gas was diluted with air. Total chamber air/DME flow was maintained at about 10 l/min.

Analytical Procedure: Gas standards and samples were analyzed with a thermal conductivity detector on a Varian Aerograph Model 700 gas chromatograph. Nitrogen, the carrier gas, flowed through a 3.5 m x 0.64 cm stainless-steel column packed with 60/80 mesh Chromosorb W with 10% SE-30. Injection port, column and detector temperatures were set at 40°, 40° and 130°C, respectively. Standards were prepared daily by quantitative dilutions of the gas, as supplied, into calibrated gas bottles containing nitrogen or air. Chamber atmospheres were sampled at approximately 30-minute intervals with a Hamilton Gastight® syringe. Both DME and oxygen were monitored from some standards (those not prepared in nitrogen) and all sample injections. Mean DME concentrations ± SD were calculated for each 6-hr. exposure.

Clinical Chemistry Protocol

Following the ninth exposure and 13th recovery day, all surviving rats were placed in metabolism cages to collect overnight (16 hr.) urine samples. After the tenth exposure and 14th recovery day blood samples were taken from the tail vein of each surviving rat. Detailed procedures are listed in the Clinical Chemistry Report (Appendix 1).

Pathology Protocol

Following the tenth exposure and 14th recovery day, 5 rats from each group were sacrificed for pathologic evaluation. At necropsy, the rats were examined grossly and selected tissues and organs were saved for histopatholic evaluation. Weights of the lungs, heart, thymus, spleen, liver, testes and kidneys were obtained from each rat. Details of pathological indices are in Pathology Raport 20-80 (Appendix 2).

Results:

Design concentrations of 5 and 1% were achieved rapidly and were uniform throughout each of the 10, 6-hour exposures.

Exposure No.	Design Level 5% Mean Concentration (%) + S.D.	Design Level 1% Mean Concentration (%) + S.D.
1	5.08 ± 0.28	1.19 ± 0.21
2	4.91 ± 0.63	1.09 ± 0.02
3	5.05 ± 0.42	1.04 ± 0.11
4	5.10 ± 0.24	1.06 ± 0.06
5	4.98 ± 0.24	1.08 ± 0.14
6	5.02 ± 0.09	1.00 ± 0.08
7	5.04 ± 0.25	1.02 ± 0.11
8	5.10 ± 0.19	1.05 ± 0.09
9	5.02 ± 0.21	1.02 ± 0.05
10	4.88 ± 0.21	1.02 ± 0.10

Overall mean concentrations were 5.02 ± 0.30% and 1.05 ± 0.11%

During exposure, no unusual behavior was observed among rats in the control and low dose (1%) groups. Rats in the high dose (5%) group were unsteady and occasionally restless. They responded very slightly or not at all to sharp taps on the chamber, while those in the control and low dose groups responded normally.

Clinical signs observed during and immediately post-exposure are outlined below.

Control Group: Occasional slight red nasal and eye discharge.
Low Dose Group: More fraquent, but slight red nasal and eye discharge, plus a moderate amount of sluggishness for a short time post-exposure, occasional slight salivation, lung noise and wet perineal area.
High Dose Group: Very frequent, but slight nasal and eye discharge; very common, moderate sluggishness post-exposure, occasional slight salivation, lung noise and wet perineal area; exophthalmos in 5/10 animals.

Body weights of the low dose group compared favorably with controls. Mean body weights of the high dose group were significantly lower (4 to 8%) than controls on test days 4 through 12, but not significantly lower during recovery (Appendix 3 and 4). Late in the first week of exposure we noticed the high dose group had more urine in the bottom of the exposure chamber. Consequently, during the second exposure week, all animals were weighed both pre- and post-exposure. The high dose group consistantly lost more weight during the 6-hour exposures (Appendix 5).

Organ/Body Weight Ratios

Mean relative organ to body weight ratios (expressed as percent) were calculated for each group, both after 10 days exposure and 14 days recovery

(Appendix 6 and 7). After 10 days exposure, liver and thymus weights were significantly lower in both test groups when compared to controls and the testes weight was significantly higher in both test groups. After 14-days recovery all organ weights were within normal limits, except the testes which remained significantly heavier in the high dose group.

Clinical Blood Chemistry and Urine Analysis

Clinical laboratory measurements (Appendix 1) demonstrated no differences between control and low dose (1%) groups after the 9th exposure or 13 days recovery.

Rats exposed to 5% DME tended to excrete a more dilute, alkaline urine than the controls and to have less urea nitrogen in blood after the tenth exposure. Three of 10 rats in this group had lower than normal total leukocyte counts but an elevation of neutrophils.

No effect on clinical laboratory measurements was found in the five remaining rats in the high dose group after 13 days recovery.

Pathology

At necropsy, no obvious gross findings were detected which were believed to be compound related. The histologic findings are shown in Pathology Report 20-80 (Appendix 2). All of the histologic changes are considered to be nonspecific and incidental or the result of intercurrent disease and not related to administration of DME.

EC50 for Anesthetic Effect

As part of the inhalation toxicity testing program, data from the acute LC50 study (HLR 847-79) and this subacute study were evaluated to determine an approximate EC50 for anesthetic effect. The EC50 seems to be in the range of 5 to 8% v/v DME in air. At 5% the animals were incapacitated (i.e., uncoordinated and unresponsive to loud noises) but not asleep. At 8% (see HLR 847-79) the animals appeared to be between delerium and light surgical anesthesia.

Summary

Subacute inhalation toxicity of DME was evaluated in male rats. Three groups of ten rats were exposed to 0, 1% or 5% DME for 6 hrs/day for 10 days. Slight signs of toxicity were observed during exposures [i.e. red nasal and eye discharge, sluggishness, salivation, lung noise, wet perineal area, exophthalmos (high dose)], which appeared to be dose related. Body weight was adversly affected at the high dose throughout the exposure period of 10 days.

Clinical blood chemistry and urine analysis demonstrated no differences between controls and the low dose group. The high dose group excreted more dilute alkaline urine and had less urea nitrogen in blood. Total leukocyte counts were lower accompanied by an elevation in neutrophils in this group after the tenth exposure. No effects were noted after 13 days recovery.

Organ to body weight ratios demonstrated a change in relative liver, thymus and testes weights after 10 days exposure at both the high and low dose.

After 14 days recovery this change in relative weight was limited to testes in the high dose group. Pathologic examination (gross and microscopic) revealed no changes considered to be compound related.

Data from the acute and subacute tests were evaluated to determine an EC50 for anesthetic effect. The EC50 in rats appears to be in the range of 5 to 8% DME v/v in air.

Conclusions: Subacute inhalation toxicity studies of DME at 1% and 5% v/v in air show that 1% DME has very low toxicity. Five percent DME has a borderline anesthetic effect and causes some discernible changes. In the absence of any pathologic lesions, the organ weights and clinical chemistry changes are difficult to evaluate.

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* Composition: 99.9+ %
20 ppm formaldehyde
40 ppm methyl formate

1 C. J. Collins, L. M. Cobb and D. A. Purser, Effects of Chronic Inhalation of Dimethyl Ether in the Rat, Toxicology, 11, 1978, 65-71.

2 L. S. Goodman and A. Gilman, The Pharmacological Basis of Therapeutics, Third Ed., The MacMillan Co., NY, 1965, p. 50.

Report by: Mavis R. Brittelli
Toxicologist

Approved by: Gerald L. Kennedy
Chief, Acute Investigations Section

MRB:jrg
Date Issued: August 11, 1980
N.B. E-5635, p. 41-64
Report No. 583-80

Triage of 8(e) Submissions

Date sent to triage: 2/5/96

NON-CAP CAP

Submission number: 13172 A

TSCA Inventory: Y N D

Study type (circle appropriate):

Group 1 - Dick Clements (1 copy total)

ECO AQUATO

Group 2 - Ernie Falke (1 copy total)

ATOX SBTOX SEN w/NEUR

Group 3 - Elizabeth Margosches (1 copy each)

STOX CTOX EPI RTOX GTOX

STOX/ONCO CTOX/ONCO IMMUNO CYTO NEUR

Other (FATE, EXPO, MET, etc.): _____

Notes:

THIS IS THE ORIGINAL 8(e) SUBMISSION; PLEASE REFILE AFTER TRIAGE DATABASE ENTRY

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entire document: 0 1 2 **pages** 1, 1st tab **pages** [redacted]

Notes:

Contractor reviewer : KPS **Date:** 12/6/95

CECATS\TRIAGE TRACKING DBASE ENTRY FORM

CECATS DATA:
Submission # 8EHQ- 0992 - 13172 SEQ. A

TYPE: INT. SUPP FLWP

SUBMITTER NAME: E. I. Dupont de Nemours and Company

INFORMATION REQUESTED: FLWP DATE:
0501 NO INFO REQUESTED
0502 INFO REQUESTED (TECH)
0503 INFO REQUESTED (VOL ACTIONS)
0504 INFO REQUESTED (REPORTING RATIONALE)
DISPOSITION:
0639 REFER TO CHEMICAL SCREENING
0678 CAP NOTICE

VOLUNTARY ACTIONS:
0401 NO ACTION REPORTED
0402 STUDIES PLANNED/UNDERWAY
0403 NOTIFICATION OF WORKERS/USERS
0404 LABEL/MSDS CHANGES
0405 PROCESS/HANDLING CHANGES
0406 APP/USE DISCONTINUED
0407 PRODUCTION DISCONTINUED
0408 CONFIDENTIAL

SUB. DATE: 09/11/92 OTS DATE: 09/22/92 CSRAD DATE: 03/24/95

CHEMICAL NAME:	CAS#
	115-10-6
	50-00-0
	107-31-3

INFORMATION TYPE:		P F C
0201	ONCO (HUMAN)	01 02 04
0202	ONCO (ANIMAL)	01 02 04
0203	CELL TRANS (IN VITRO)	01 02 04
0204	MUTA (IN VITRO)	01 02 04
0205	MUTA (IN VIVO)	01 02 04
0206	REPRO/TERATO (HUMAN)	01 02 04
0207	REPRO/TERATO (ANIMAL)	01 02 04
0208	NEURO (HUMAN)	01 02 04
0209	NEURO (ANIMAL)	01 02 04
0210	ACUTE TOX. (HUMAN)	01 02 04
0211	CHR. TOX. (HUMAN)	01 02 04
0212	ACUTE TOX. (ANIMAL)	01 02 04
0213	SUB ACUTE TOX (ANIMAL)	01 02 04
0214	SUB CHRONIC TOX (ANIMAL)	01 02 04
0215	CHRONIC TOX (ANIMAL)	01 02 04

INFORMATION TYPE:		P F C
0216	EPI/CLIN	01 02 04
0217	HUMAN EXPOS (PROD CONTAM)	01 02 04
0218	HUMAN EXPOS (ACCIDENTAL)	01 02 04
0219	HUMAN EXPOS (MONITORING)	01 02 04
0220	ECO/AQUA TOX	01 02 04
0221	ENV. OCCC/REL/FATE	01 02 04
0222	EMER INCI OF ENV CONTAM	01 02 04
0223	RESPONSE REQEST DELAY	01 02 04
0224	PROD/COMP/CHEM ID	01 02 04
0225	REPORTING RATIONALE	01 02 04
0226	CONFIDENTIAL	01 02 04
0227	ALLERG (HUMAN)	01 02 04
0228	ALLERG (ANIMAL)	01 02 04
0239	METAB/PHARMACO (ANIMAL)	01 02 04
0240	METAB/PHARMACO (HUMAN)	01 02 04

INFORMATION TYPE:		P F C
0241	IMMUNO (ANIMAL)	01 02 04
0242	IMMUNO (HUMAN)	01 02 04
0243	CHEM/PHYS PROP	01 02 04
0244	CLASTO (IN VITRO)	01 02 04
0245	CLASTO (ANIMAL)	01 02 04
0246	CLASTO (HUMAN)	01 02 04
0247	DNA DAM/REPAIR	01 02 04
0248	PROD/USE/PROC	01 02 04
0251	MSDS	01 02 04
0299	OTHER	01 02 04

TRIAGE DATA:	NON-CBI INVENTORY	ONGOING REVIEW	SPECIES	TOXICOLOGICAL CONCERN:	USE:	PRODUCTION:
	YES	YES (DROP/REFER)	RAT	LOW Subacute Inhalation Toxicity		
CAS SR	NO	NO (CONTINUE)		MED		
	DETERMINE	REFER		HIGH		

COMMENTS

#13172A
L

Subacute inhalation toxicity is of low concern based on no mortality in rats (10/dose) exposed to 1 and 5% concentrations (100 and 500 ppm) for 6 hours/day, 5 days/week for 2 weeks. Clinical signs included moderate sluggishness (both doses).